

EXHIBIT “O”

Expert Report
Confidential

November 27, 2017

Re: *Pagan v. New York City, et al.*

EXPERT REPORT OF LARISSA LASKOWSKI, DO

At issue here is the cause of death of Mario Ocasio, the plaintiff's son, and whether the actions of New York Police Department (NYPD) officers and Fire Department of New York (FDNY) Emergency Medical Services (EMS) providers caused or contributed to his death. Namely, it is alleged that Mr. Ocasio died as a result of forceful restraint and/or electrical control device (ECD) application by NYPD officers, in conjunction with the intramuscular administration of midazolam 10mg by EMS paramedics.

Regarding the death of Mario Ocasio, I reviewed the following items:

- Amended Complaint
- 911 Recordings
- Mrs. Lloyd's Statement
- Deposition Transcript of Paramedic Bree Brown-Rose
- EMS Providers' Statements to Investigators
- I/Net Dispatcher Event Chronology D15060804988 (aka Sprint report)
- FDNY Prehospital Care Report Summary
- NYPD Officers' Statements to Investigators
- Allan Pavilion Medical Records
- Bronx Lebanon Hospital Comprehensive Care Center Clinic Records
- St. Barnabas Hospital Medical Records
- Plaintiff Adela Pagan's Amended Designation of Experts and Disclosure of Expert Testimony Pursuant to Rule 26(a)(2) of the Federal Rules of Civil Procedure
- TASER® Handheld CEW Warnings, Instructions, and Information: Law Enforcement
- TASER® Training Bulletin 15.0 Regarding Medical Research Update and Revised Warnings
- OCME Autopsy Report and file

To address the plaintiff's claim, my report (below) consists of the following:

- Brief on the decedent's past medical history
- Description of the circumstances prior to and immediately after the decedent's death, with attention to medical details
- Review of the autopsy report, with attention to documented trauma, Taser barb findings, and forensic toxicology
- Description of K2 (synthetic cannabinoids) and AB-CHMINACA, including a brief literature review

- Comment on the expert report of Ibrahim Fatiha, with attention to the opinions about K2 and ECD application contributing to Mr. Ocasio's death
- Case conclusion

In brief, based on the material provided to me I understand the facts to be as follows: Mario Ocasio was a 51-year-old man with a past medical history of HIV/AIDS, avascular necrosis, asthma, polysubstance abuse (marijuana, cocaine), hypertriglyceridemia, and hepatomegaly.

On the morning of June 8, 2015, Mr. Ocasio was home with his common-law wife, Mrs. Lloyd, when he told her he was "going to get something". He returned shortly thereafter, and was acting strangely, "agitated and threatening her." He picked up a pair of scissors and came towards her, so she locked herself in a bedroom and called 911. NYPD officers report that upon arrival, they found Mr. Ocasio lying on the floor, not responding appropriately to commands and resisting restraint. NYPD officers report that early attempts to physically restrain Mr. Ocasio failed, so they used a Taser or electrical control device (ECD), shocking him in the back, without any immediate change in his behavior. Subsequent to being tased, Mr. Ocasio was handcuffed behind his back. Upon EMS arrival, FDNY EMS providers report that the patient was "awake but disoriented" and not responding appropriately to commands. FDNY EMS paramedic Bree Brown-Rosa reports that upon arrival, Mr. Ocasio was awake, restrained by two police officers on his side with two Taser darts in his mid-back and handcuffed from behind. Per Paramedic Brown-Rosa's deposition, upon examining Mr. Ocasio, she noted that he was disoriented, but made eye contact, and was "shouting that he was God, and saying things in Spanish, and trying to move around." The patient was further examined and placed on a continuous monitor. Vital signs and ECG were documented as follows:

- 8:34 am: blood pressure, 120/94 mm Hg; heart rate, 134; respiratory rate, 18; oxygen saturation, 97% on room air.
- 8:36 am: 12-lead ECG revealed sinus tachycardia.

To control, sedate and transport Mr. Ocasio down three flights of stairs to the ambulance (and subsequently to the hospital), midazolam 10mg intramuscularly (IM) was administered for chemical restraint, according to NYPD/EMS pre-hospital protocol. During this period, vital signs were obtained and oxygen was administered and documented as follows:

- 8:40 am: blood pressure, 120/98 mm Hg; heart rate, 136; respiratory rate, 18; oxygen saturation, 97% on room air; and blood glucose, 134 mg/dl.
- 8:42 am: 100% oxygen was administered via non-rebreather (NRB) mask
- 8:44 am: blood pressure, 124/98 mm Hg; heart rate, 132; respiratory rate, 16; oxygen saturation, 97% on room air.
- 8:47 am: oxygen saturation, 100%

The patient was transferred to a scoop stretcher and transported down three flights of stairs to the street. Per EMS providers' report, on the street the patient was noted to be breathing spontaneously. After being placed in the ambulance, and "in the process of turning [the patient] from [right lateral recumbent] to [left lateral recumbent], it was

noticed that [the patient] was apneic and pulseless.” The scoop stretcher, restraints, and handcuffs were immediately removed and cardiopulmonary resuscitation (CPR) was initiated. Advanced Cardiac Life Support (ACLS) protocol commenced and included continuous CPR, endotracheal intubation, and intravenous medication administration of epinephrine, naloxone, and sodium bicarbonate, without response. The cardiac rhythms documented during the cardiac arrest were asystole, pulseless ventricular tachycardia (v-tach) twice (for which the patient was defibrillated twice), and asystole. According to medical records from the Allen Pavilion, the patient arrived in cardiac arrest, where ACLS protocol was continued. A point of care blood gas was performed and revealed pH 6.9 with bicarbonate 18 mEq/L. Bedside echo showed no cardiac activity and the patient was pronounced dead at 9:50 am. No additional laboratory or diagnostic testing was performed.

The autopsy report by medical examiner Dr. John A. Hayes, concluded the following:

- “Cause of death: Cardiac arrhythmia during excited delirium due to acute intoxication by synthetic cannabinoid (AB-CHMINACA)
- “Contributory: Hypertensive cardiovascular disease; chronic substance abuse
- “Manner of Death: Accident”

Regarding Taser findings, the autopsy report concluded:

- “Status post tasering: two Taser barbs recovered from midback”

Regarding traumatic injuries, the autopsy report concluded:

- “Multiple cutaneous abrasions and contusions, superficial: patterned injuries of wrists consistent with handcuff application
- “No visceral injuries or fractures
- “No evidence of neck or chest compression”

Regarding forensic toxicology results:

- “Post-mortem toxicology positive for synthetic cannabinoid (AB-CHMINACA)”

Upon thorough review of the complete autopsy report and accompanying photographs, I have duly noted the following: 1) Taser barbs were found in the patient’s mid-back; 2) only superficial traumatic injuries were present; 3) forensic toxicology testing confirmed the presence of the synthetic cannabinoid AB-CHMINACA. Notably absent from the autopsy report is any evidence to suggest the patient sustained lethal traumatic injuries. In summary, and in line with Dr. Hayes findings, I have concluded that Mr. Ocasio’s death was a result of the effects of the synthetic cannabinoid AB-CHMINACA.

Relevant to this case is an understanding of the deleterious and occasionally lethal effects of synthetic cannabinoids (or synthetic cannabinoid receptor analogues (SCRAs)) such as AB-CHMINACA. Known on the street as “K2” or “Spice”, synthetic cannabinoids are a novel class of designer drugs, which have emerged in recent years as a cheap alternative to marijuana. Synthetic cannabinoids were first synthesized as early as the 1960s, for biomedical research purposes. Beginning in the late 2000s, these agents were replicated by street chemists in clandestine labs, sprayed onto dried plant material, and packaged with brand names like K2 and Spice. To date, there are over 100 documented synthetic cannabinoids, many of which have never before been seen, and therefore lack any controlled animal or human data to describe their clinical effects or safety profile. AB-

CHMINACA, for example, was not reported in the scientific literature prior to its appearance on the illicit drug market.

In the brain, synthetic cannabinoids bind to the same cannabinoid receptors as traditional cannabinoids, such as delta-9-THC, the cannabinoid found in marijuana. However, synthetic cannabinoids and their metabolites bind to these receptors with greater potency and binding affinity, and additionally bind to other non-cannabinoid receptors, resulting in the wide and often unpredictable range of clinical effects seen. Based on anecdotal evidence and case report data, onset of clinical effects occurs within minutes and typically last hours, but can range from minutes to days. Of note, pharmacokinetic and pharmacodynamic profiles of most synthetic cannabinoids is largely unstudied and unknown (1,2). As there is no quality control and thus no way of knowing which substance is present in a particular batch, the effect on individuals who smoke K2 is also largely unpredictable. With regard to forensic tests (which can now detect over 100 unique SCAs), as new substances emerge daily, failed detection often occurs.

Case reports documenting the severe and occasionally lethal adverse effects of synthetic cannabinoids abound. According to a comprehensive review that compiled data through December 2014, reports on over 4000 cases have been documented in the medical literature, including a conservative estimate of between 22 to 27 deaths (3). Since then, at least 24 additional deaths linked to synthetic cannabinoids have been reported (4). (Of note, these numbers only reflect those cases documented in medical journals, conferences, or abstracts. This is just a small fraction of the total number of individuals who have presented to healthcare with adverse effects and/or death secondary to synthetic cannabinoids). Among documented cases, the following major complications have been described: nausea, vomiting, hyperemesis or uncontrollable vomiting, cardiovascular events (dysrhythmia, heart attack, stroke, cardiac arrest), organ failure (kidney, liver), seizures, psychiatric presentations (agitation, first episode psychosis, anxiety, paranoia, self-harm/suicidal ideation), sedation, coma, and death (2,3).

A case series published in 2015, described 35 patients who presented to a single Florida hospital over a 12-day period, with symptoms ranging from altered mental status, hallucinations, acute delirium, and seizures, to coma and bradypnea (slow breathing) requiring ventilatory support and ICU-admission (5). Laboratory analysis confirmed the presence of AB-CHMINACA in 15 of 21 patients whose blood or urine samples were available for testing.

In the expert report submitted by Ibrahim Fatiha, it was stated that:

“It is absolutely no doubt that K2 could ever be a contributing factor to the victim’s death due to no only the fact that there is no history or finding of Renal failure or signs and symptoms of cardiac issues at the time of consumption... he only exhibited some type of misbehavior which could be resolved within half an hour more or less... K2 affects the central nervous system (CNS) which evidently led to abnormal behavior such as hallucination (mild) and without any aggression... it has absolutely no

effect on the cardiovascular system... it is only could have an effect on the cardiovascular system if he would've had kidney failure for a long time (chronic)...

To address to the above statement, the presence or absence of pre-existing renal failure plays no role in the acute presentation of synthetic cannabinoid exposure. Renal failure has been documented to occur *after* exposure, however we do not know if the patient developed this complication, as the patient expired before laboratory tests could be ordered by the receiving hospital. Mr. Ocasio, did, however exhibit "cardiac issues" secondary to exposure, as manifest by an elevated heart rate (tachycardia). In fact, agitation and tachycardia are the two most common findings documented after synthetic cannabinoid exposure (3).

Ibrahim Fatiha also submitted in his expert report that:

"The Federal government has restricted all physicians from prescribing any CNS depressant to anyone who is taking even the lowest dose of benzodiazepines because it really slows down the breathing pattern and leads to labored breathing (as Ms. Rosa Brown alleged that his breathing slowed down but not his heart rate)... that means he had labored breathing and made his heart convert to further damage."

Contrary to what is suggested in the above statement, neither federal government restriction nor contraindications exist to administering central nervous system (CNS) depressants to patients who are prescribed, and may be taking, benzodiazepines. A number of CNS depressants exist, including midazolam (Versed), the benzodiazepine administered by EMS to Mr. Ocasio. As an emergency physician, I regularly administer CNS depressants to patients who may be taking "even the lowest dose of benzodiazepines." In fact, patients who regularly take benzodiazepines often become tolerant to these drugs, and often require higher doses to achieve sedation.

Goals of treatment in administering intravenous benzodiazepines include: decreased agitation, sedation, decreased sympathetic output (i.e. lower blood pressure and heart rate). As mentioned by Mr. Fatiha above, an anticipated side effect is decreased respiration, especially when given in high doses. In order to rapidly and safely transport the patient to healthcare, EMS workers administered midazolam 10mg intramuscularly (IM) to Mr. Ocasio according to protocol. Based on consensus by an American College of Emergency Physicians Task Force, the pre-hospital management of Mr. Ocasio's Excited Delirium Syndrome (ExDS) also fell within recommended guidelines (6). Per EMS documentation, the patient's oxygen saturation was assessed after drug administration, and supplemental oxygen was applied, both mitigating the effects of a slower respiratory rate.

The final paragraphs of Mr. Fatiha's expert report appear below. Of note, this text also appears verbatim in an article published by Zipes, and has been cut and pasted from this article by me to highlight the similarities (7):

Causality

A temporal association alone does not prove causality. However, when the following exist, in my opinion, a causal relationship between the TASER X26 ECD and cardiac arrest in humans is established: (1) known causal mechanism (cardiac capture at rapid rates), (2) temporal association with loss of consciousness and subsequent cardiac arrest (TASER shock precedes both), (3) recorded VF (or asystole if a prolonged interval until first ECG; biological mechanism), (4) TASER shock(s) with 1 or both chest barbs near the heart (required for cardiac capture), (5) no other plausible alternative explanation (normal heart or underlying heart disease/drugs, if present, unlikely to cause VF at that precise time), and (6) similar cases in the literature (see above).

Conclusions and Recommendations

The animal and clinical data clearly support the conclusion that a TASER X26 shock can produce VF in humans by the mechanisms elaborated above. Although the risk may be low, its number cannot be known without universal record keeping and the creation of a national database. Because of this risk, it has been suggested that law-enforcement experts reassess ECD use to maintain a balance of safety for subjects and officers while still achieving the goal of maintaining law and order.⁵⁵ In this regard, the Cincinnati Police Department has revised its use-of-force policy to ban TASER chest shots except in self-defense or the defense of another.⁵⁶

The use of TASERS may be increasing. A recent *Guardian* article indicated that the deployment of TASER weapons has more than doubled in England and Wales, from ≈3500 in 2009 to 14 500 in 2010 and 2011.⁵⁷ In addition, a new TASER ECD, the X2, capable of shooting 2 cartridges, has been tested in 4 pigs exposed to 5-second shocks; it produced cardiac capture in 17 of 71 exposures (24%) at heart rates of 206 to 313 bpm compared with X26 capture in 45 of 71 exposures (63%) at heart rates of 180 to 313 bpm.⁵⁸ No pig developed VF. The authors concluded that the “transcardiac” pathway was less important for capture than the proximity of the dart to the heart.

I think ECD manufacturers should undertake an educational campaign to make all ECD users aware of the VF risk. Educational material should stress avoiding chest shots if possible and should warn against repeated or long trigger pulls. However, it is clear that a single 5-second shock can induce VF. A user should be judicious with ECD deployment and treat it with the same level of respect as a firearm, suspect cardiac arrest in any individual who becomes unresponsive after a shock, quickly call for medical support, and be prepared to resuscitate, including using an

automated external defibrillator if needed. A national database should be mandated.

I believe Mr. Fatiha plagiarized this excerpt from Zipes paper to support the claim that Mr. Ocasio's death was caused by Taser/ECD application. Both this article and another by Dr. Zipes (the latter referenced in Mr. Fatiha's expert report) provide evidence that ECDs can cause cardiac arrest in humans (7,8). Specifically, both articles describe a series of eight cases (collected as part of litigation proceedings) of sudden cardiac arrest after the administration of ECD shocks from the TASER X26 device. In all eight cases, shocks were received in the anterior chest overlying the heart, and in all cases, loss of consciousness (indicating cardiac arrest) occurred immediately or within seconds of Taser administration. Mr. Ocasio, in contrast, received ECD shocks to the mid-back with no reported change in his behavior or level of consciousness after the Taser was applied. In fact, after Mr. Ocasio received ECD shocks, EMS arrived on scene, took multiple sets of vital sign measurements (spanning 10 minutes), performed a 12-lead ECG, transported him manually down three flights of stairs, and loaded him into the ambulance, before he developed cardiac arrest. While the true incidence of ECD-induced cardiac arrest is unknown, risk has been postulated at 1:100,000 applications (9). The evidence provided by the two Zipes articles (one referenced, the other plagiarized), strongly counter the hypothesis that Taser shocks caused or contributed to Mr. Ocasio's cardiac arrest.

After thorough review of the available evidence, it is my opinion, to a degree of reasonable medical certainty, that Mr. Ocasio died as a result of the effects of the synthetic cannabinoid receptor analogue AB-CHMINACA, namely, agitated or excited delirium and sympathomimetic excess causing dysrhythmia and cardiac arrest. Furthermore, there is no evidence in any of the reports to suggest that the actions of police and EMS caused or contributed to his death. Lastly, if, contrary to the officers' statements, Mr. Ocasio was tased after he was handcuffed and/or was not actively resisting NYPD officers, my opinion about the cause of his death would not change.

Sincerely,

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Emergency Medicine | Medical Toxicology

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